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Related Articles
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Attenuation and augmentation of ischaemia-related neuronal deat by tumour necrosis factor-alpha in vitro.

Wilde GJ, Pringle AK, Sundstrom LE, Mann DA, Iannotti F.

Eur J Neurosci. 2000 Nov;12(11):3863-70.

Department of Clinical Neurological Sciences, LF73B, Level F, South Academic Block, Mailpoint 806, University of Southampton, Southampton General Hospital, Tremona Road, Southampton SO16 6YD, UK.

Upregulation of the pro-inflammatory cytokine tumour necrosis factor alpha (TNF) occurs rapidly in the brain following ischaemia, although is unclear whether this represents a neurotoxic or neuroprotective response. We have investigated whether TNF has different actions in the pre- and postischaemic periods in a tissue culture model of cerebra ischaemia. Organotypic hippocampal slice cultures were prepared fron 8-10-day-old rats and maintained in vitro for 14 days. Neuronal damag was induced by either 1 h oxygen-glucose deprivation or 3 h exposure to NMDA or the superoxide generator duroquinone, and assessed after 24 h by propidium iodide fluorescence. TNF pretreatment was neuroprotective against both oxygen-glucose deprivation and duroquinone. This effect was associated with an activation of the transcription factor NFkappaB and upregulation of manganese superoxide dismutase, and was prevented by a free radical scavenger. When addition of TNF was delayed until the postinsult period, an exacerbation of neurotoxicity occurred, which was also prevented by a free radical scavenger. The actions of TNF are determined by whether TNF is present before or after an ischaemia-related insult. Both actions are mediated through the production of free radicals, and the response to TNF is determined by whether a cell is metabolically competent to respond by synthesis of antioxidant defences.

MeSH Terms:
- Animal
- Benzoquinones/pharmacology
- Cell Death
- Free Radical Scavengers/pharmacology

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Differential vulnerability of the CA1 and CA3 subfields of the hippocampus to superoxide and hydroxyl radicals in vitro.

Wilde GJ, Pringle AK, Wright P, Iannotti F.

J Neurochem. 1997 Aug;69(2):883-6.

Department of Clinical Neurological Sciences, University of Southampton, Southampton General Hospital, England, U.K.

The relative roles of the superoxide and hydroxyl radicals in oxidative stress-induced neuronal damage were investigated using organotypic hippocampal slice cultures. Cultures exposed to 100 microM duroquinone, a superoxide-generating compound, for 3 h developed CA1-selective lesions over a period of 24 h. The damage accounted for approximately 64% of the CA1 subfield, whereas CA3 showed just 6% damage, a pattern of damage comparable to that observed following hypoxia/ischaemia. Duroquinone-induced damage was attenuated by a spin-trap agent. In contrast, hydroxyl radical-mediated damage, generated by exposure to 30 microM ferrous sulphate for 1 h, resulted in a CA3-dominant lesion. The damage developed over 24 h, similar to that observed with duroquinone, but with approximately 45% damage in CA3 compared with only 7% in CA1. These data demonstrate a selective vulnerability of the CA1 pyramidal neurones to superoxide-induced damage and suggest that of the free radicals generated following hypoxia/ischaemia, superoxide, rather than hydroxyl radical, is instrumental in producing neuronal damage.

MeSH Terms:

- Animal
- Benzenesulfonates/pharmacology
- Benzoquinones/pharmacology
- Brain Ischemia
- Cell Death
- Ferrous Compounds/pharmacology
- Hippocampus/*drug effects
- Hydroxyl Radical/ pharmacology
- Hypoxia, Brain
- Neurons/drug effects

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Related Articles

Binding of botulinum type Cl, D and E neurotoxins to neuronal cel lines and synaptosomes.

Yokosawa N, Tsuzuki K, Syuto B, Fujii N, Kimura K, Oguma K.

Toxicon. 1991;29(2):261-4.

Department of Microbiology, Sapporo Medical College, Japan.

Clostridium botulinum 125I-labeled Cl neurotoxin bound to NG108 hybridoma cell line. Unlabeled type Cl neurotoxin inhibited the bindin of the labeled Cl toxin but neither types D nor E toxin. 125I-labeled type D neurotoxin bound to rat brain synaptosomes but did not bind to NG108 cells. It is suggested that receptors for types C and D or E toxir on neuronal cell membranes are different.

MeSH Terms:
- Animal
- Binding, Competitive
- Botulinum Toxins/*metabolism
- Cell Line
- Human
- Mice
- Neuroblastoma/metabolism
- Neurons/*metabolism
- Rats
- Synaptosomes/*metabolism
- Tumor Cells, Cultured

Substances:
- 0 (Botulinum Toxins)

PMID: 2048142 [PubMed - indexed for MEDLINE]

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Related Articles

Binding of Clostridium botulinum type C neurotoxin to different neuroblastoma cell lines.

Yokosawa N, Kurokawa Y, Tsuzuki K, Syuto B, Fujii N, Kimura K, Oguma K.

Infect Immun. 1989 Jan;57(1):272-7.

Erratum in:
- Infect Immun 1989 Jul;57(7):2265. PMID: 0

Department of Microbiology, Sapporo Medical College, Japan.

Binding of type C neurotoxin (C1 toxin) from Clostridium botulinum (strain Stockholm) to neuroblastoma cell lines was studied by using biotinylated anti-toxin antibody and avidin-biotinylated peroxidase complex. The neurotoxin bound with high efficiency to mouse neuroblastoma (NS-20Y and NIE-115) cells and to hybridomas of rat glioblastoma and mouse neuroblastoma (NG108-C15) cells. The toxin bound little to human neuroblastoma, rat astrocytoma, and nonneural cell lines. Binding of the neurotoxin to NG108-C15 cells was inhibited by gangliosides (GT1b and GM1) and by monoclonal antibodies (CA-12 and C-9), although inhibition was not complete. Sequential preincubation of C1 toxin with GT1b and CA-12 caused complete inhibition. A Scatchard plot of binding of 125I-labeled C1 toxin to NG108-C15 cells showed a hyperbolic curve. Monoclonal antibody CA-12 but not C-9 neutralized the lethal activity of the toxin toward mice. Only C-9 clearly inhibited toxin binding to GT1b. These results suggest that NG108-C15 cells have at least two kinds of receptors for C1 toxin. From the results of binding tests with neuraminidase-, pronase-, and trypsin-treated NG108-C15 cells, the chemical nature of the high-affinity site was presumed to be a glycoprotein containing sialic acid. GT1b may have an important role in low-affinity sites.

MeSH Terms:
- Animal
- Antibodies, Monoclonal
- Binding, Competitive